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Review

A look at common gastrointestinal system diseases: current review

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ABSTRACT

The gastrointestinal system consists of the esophagus, stomach, small intestine, large intestine, and rectum. Although they are called accessory organs, the liver, pancreas, and gallbladder are also evaluated in this system with their vital features. Gastrointestinal system disorders (GISD) are observed quite frequently in society. Considering the morbidity and mortality they cause, effective treatment methods become crucial. Situations such as patients' non-compliance with nutritional recommendations and the inadequacy of pharmacological and surgical methods at some points make different perspectives and treatment methods more valuable. From this point of view, we will try to examine the GISD that causes muscle loss and the physiotherapy modalities that can be applied to these patients in light of the current literature.

Keywords: Gastrointestinal system, physiotherapy, role

INTRODUCTION

The gastrointestinal system (GIS) consists of the esophagus, stomach, small intestine, large intestine, and rectum. Although they are called accessory organs, the liver, pancreas, and gallbladder are also evaluated in this system with their vital features. Gastrointestinal system disorders (GISD) are observed quite frequently in society. Considering the morbidity and mortality they cause, effective treatment methods become crucial. Situations such as patients' non-compliance with nutritional recommendations and the inadequacy of pharmacological and surgical methods at some points make different perspectives and treatment methods more valuable. From this point of view, we will report mostly seen GIS diseses in short.[1-3]

PROTEIN LOSING ENTEROPATHIES

Protein-losing enteropathy (PLE) is a clinical condition that develops as a result of excessive protein loss from the gastrointestinal tract, characterized by low protein in the blood, edema, and fluid accumulation in the lung and heart membranes in some patients. However, to use the term PLE, first, long-term malnutrition, urinary protein loss, and protein production disorders due to liver diseases must be excluded.[1]

Under normal conditions, the contribution of the intestinal mucosa to protein degradation is quite insignificant. However, this rate increases considerably in PLE. The human body can compensate for low protein losses by increasing protein synthesis activities, but when the intestinal loss rate reaches 60%, it is insufficient.[2] Many diseases can cause this clinical condition. From this point of view, it would be more logical to briefly talk about the pathophysiological mechanisms of intestinal-induced protein loss, rather than listing individual diseases.[1,3]

Injury of the intestinal surface and secretion of inflammatory fluid with high protein content due to causes such as inflammatory bowel diseases, infective agents, cancers. Conditions where only permeability increases without erosion or ulceration on the mucosal surface, such as Giardia infection, rheumatic diseases, celiac disease, hypertrophic gastric mucosa. Obstruction of protein-carrying intestinal lymphatics due to either congenital defects or causes such as cancers, granulomatous diseases, and heart failure.[3,4]

Some diseases may cause PLE through several of the above-mentioned mechanisms. Considering that PLE is the result, not the cause, the clinical findings will vary depending on the underlying disease. For example, a factor such as C. Difficile will apply to the hospital with abdominal pain, bloody diarrhea, and fever, while a patient with heart failure will present with shortness of breath and decreased effort capacity. On the other hand, a patient with a decreased circulating immunoglobulin level will have frequent infections. The decrease in the amount of protein will reduce the intravascular oncotic pressure, which will cause fluid leakage from the intravascular to the outside. Fluid accumulation in the intercellular space will appear as edema. Although edema is expected in all patients, it is not that common.[4]

So, are there any laboratory tests to support our diagnosis of suspected patients? Undoubtedly yes. Serum albumin, immunoglobulin, cholesterol, and transferrin levels may be low. In addition, alpha-1 antitrypsin molecules synthesized in the liver can be used for diagnostic purposes. This molecule is excreted in a certain amount with feces under normal conditions and is resistant to digestion. If 24-hour alpha-1 antitrypsin clearance is calculated in the collected stool and

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found to be higher than the expected level, this indicates an intestinal-mediated loss. Labeled macromolecules can also be used for this purpose.[5]

After concluding that the patient has PLE, a more detailed examination should be performed for the underlying cause. Although it varies according to the physician's opinion and experience; kidney and liver function tests, celiac disease serology, stool parasite, and microscopic examination, C. difficile toxin in stool, serum vitamin D, and cholesterol level are the recommended tests. In addition; Abdominal imaging methods can be performed for inflammatory bowel diseases, lymphoma, enlargement, and obstruction of the lymphatics. Endoscopic methods that allow direct visualization of the mucosa and obtaining pathological samples may also be preferred. Echocardiography in patients with suspected heart disease, and specific antibodies in rheumatic diseases are other tests that can be preferred.[5-7]

After the diagnosis of PLE is confirmed, the patient's nutrition should be regulated without losing time. Studies support a diet low in fat, rich in protein, and medium-chain fatty acids. Long-chain fatty acids should be avoided. Because these molecules, unlike medium-chain fatty acids, are absorbed through protein. In these patients, diets containing 3 g of protein per kilogram should be preferred. In addition, research should be continued to detect the underlying disease and treatment should be started for it.[7]

INFLAMMATORY BOWEL DISEASES

Inflammatory bowel diseases (IBD) are thought to develop as a result of the impaired immune response against intestinal microflora and include ulcerative colitis (UC) and Crohn's disease (CD).[8] UC is limited to the colonic mucosa, progresses with recurrent attacks, starts from the rectum, and spreads proximally. CD affects the entire gastrointestinal tract from the mouth to the anus. In both diseases, systems such as eyes, joints, and skin may also be affected, as well as intestinal involvement. Its incidence is approximately 12/100,000. Both diseases peak twice, at young and advanced ages. UC mostly affects men and CD affects women more.[9] The probability of having IBD in first-degree relatives of patients has increased approximately thirty times compared to the general population. Likewise, considering that this rate is as high as 35% in homozygous twins, it is obvious that the disease has a serious genetic predisposition.[10] How IBD develops is still unknown. However, in individuals with genetic predisposition, an exaggerated immune response against intestinal flora with the effect of environmental factors is one of the most plausible views. Inadequate intake of breast milk, smoking, excessive consumption of animal foods, and a diet poor in omega-3 fatty acids are the environmental factors that are blamed.[11] With the increase in intestinal permeability, the emergence of an immune response against antigens that cannot pass the mucosal barrier under normal conditions is another mechanism that is held responsible for the development process of the disease. In addition, studies have shown that the balance between Th1 and Th2 cells, which are vital in controlling inflammation, is impaired in these patients.[11-13]

UC begins in the rectum and spreads proximally. Involvement is limited to the mucosa. Areas without involvement are not observed between the intestinal segments. The rectum region is most affected, but the entire large intestine may be affected in one-sixth of patients. Patients usually present with abdominal pain, bloody-mucous diarrhea, and weight loss. These symptoms are often observed as intermittent exacerbations or may be less persistent. One-twentieth of the patients may experience 20-30 defecations per day, fever, and abdominal pain in the form of cramps. There are no physical examination findings specific to UC. During the attack periods, abdominal tenderness and fever are observed. Rectal examination reveals fresh bleeding or bloody stools.[12] In the blood tests performed during this period, an increase in the erythrocyte sedimentation rate, C-reactive protein level and white blood cell level, and anemia may be observed. To exclude other causes, stool culture should be performed together with stool microscopic and parasitological examination. Undoubtedly, endoscopic imaging methods are very valuable in IBD, as they allow both direct visualizations of lesions and biopsy in terms of differential diagnosis. In periods of exacerbation, the mucosa is edematous and prone to bleeding; ulcers and false polyp structures are detected in the intestine. As the disease progresses, the large intestinal mucosa acquires a granular appearance. Contrast-enhanced radiological examinations show a sawtooth appearance in the exacerbation period and a lead pipe appearance in the chronic period. Undesirable bowel-related conditions such as severe bleeding, toxic megacolon, perforation, and obstruction may develop due to UC.[13] In addition, extra-intestinal clinical manifestations such as uveitis, iridocyclitis, pyoderma gangrenosum, erythema nodosum, and ankylosing spondylitis may accompany.[14] In these patients, the probability of developing colon cancer in ten years is one in twenty. In addition, progressive fibrosis of the biliary tract is another feared clinical condition.

The CD is mostly located in the small intestine, the large intestine is less affected. In about half of the cases, both organs are involved. Especially the last part of the ileum is affected in almost all patients. Unlike UC, it first starts with superficial ulcers, then these ulcers merge, but there are intact mucosal areas in between. Because of this segmentary involvement, a cobblestone appearance occurs in the mucosa. Due to the complete involvement of the intestinal wall, thickening of the adjacent mesentery tissue and enlargement of the lymph nodes are observed. Granulomatous inflammation is one of the hallmarks of CD. Fistulas may develop between adjacent bowel segments and between the bowel and other organs and skin. Unlike UC, bloody stools are not expected. Patients mostly present with abdominal pain, fever, and diarrhea. In laboratory examination, an increase in acute phase reactants, increased fat excretion in the stool due to malabsorption, and iron, zinc, and vitamin deficiencies can be detected. In imaging studies, narrowing of the intestinal lumen, wall thickening, fistulas, and abscesses can be observed. Aphthous ulcers are detected in the endoscopic examination and the diagnosis is confirmed by the pathological samples taken. Unlike UC, it progresses with granulomatous inflammation. Abscess, obstruction, fistula, and perforation may develop due to CD.[12,13,15]

In both diseases, diet is primarily regulated. Fluid, mineral, and vitamin supplements are provided. Raw fruits and vegetables, spicy and fatty foods are avoided in the diet. Antidiarrheal and antispasmodic drugs, antibiotics for CH can be used. 5-aminosalicylic acid derivatives, steroids, and classical treatment agents that suppress the immune system are widely used in the treatment. In addition, target-specific monoclonal antibodies have recently been used.[13,16]

LIVER DISEASES

Non-alcoholic Fatty Liver Disease

Non-alcoholic fatty liver disease (NAFLD) is used to express fatty liver. However, secondary causes of fatty liver such as alcohol use should be excluded. The concept of non-alcoholic fatty liver disease (NAFLD) is used only for patients with fatty liver, and the concept of non-alcoholic steatohepatitis (NASH) is used more for conditions accompanied by inflammation. It is the most common liver disease, especially in developed societies and often accompanies diabetes, obesity, hyperlipidemia, and metabolic syndrome. Although its distribution and prevalence vary in different studies, it is thought to affect approximately one-fifth of the population.[17,18]

Unfortunately, the development process of the disease has not been fully elucidated. However, the most accepted theory is insulin resistance. The presence of metabolic syndrome in most of the patients supports this opinion. Fat accumulation occurs in the liver because of excessive absorption of free fatty acids, increased destruction in surrounding tissues, decreased intracellular use, and increased conversion of other nutrients to fatty acids. Insulin resistance lies at the root of these events. Free radicals are formed because of the peroxidation of these accumulated oils. These radicals are reduced through vitamin C, vitamin E, and glutathione, which are among the treatment methods. However, exceeding the capacity causes liver damage. Fatty liver is also observed in hemochromatosis disease, which occurs because of a genetic disorder and is characterized by iron accumulation in the body. Based on this, it is thought that iron has a role in the etiology of NAFLD. In addition, a relationship between NASH and resistance to leptin molecules produced in adipose tissue in the central nervous system has been demonstrated. There is an inverse relationship between the level of adiponectin, another molecule released from adipose tissue, and fatty liver. The opinion that intestinal flora triggers fatty liver and fibrosis in some patients is extremely interesting. Recent studies support this theory. Stellate cells in the perisinusoidal area are responsible for the fibrosis that occurs in the advanced stages of the disease.[13,18-20]

Despite being so common and having serious consequences, its clinical manifestations are rather faint. Complaints are often attributed to other causes, such as weakness, malaise, and discomfort in the upper abdomen. Patients are usually detected incidentally in tests performed for other reasons, and a mild liver enlargement may be observed in approximately one-fifth of them. Laboratory investigations may reveal moderate elevation of transaminases. However, there is no correlation between elevated transaminase levels and liver inflammation. Likewise, a normal range of transaminase levels does not exclude inflammation.[20-22]

For diagnosis, fatty liver should be demonstrated by biopsy or imaging methods, alcohol use and other genetic and viral chronic liver diseases should be excluded. NASH and NAFLD can only be distinguished by pathological examination, but since biopsy is risky and the treatment approach does not change much, this option should be reserved for risky patients only. Patients with signs and symptoms of cirrhosis, high serum ferritin levels, and patients with metabolic syndrome over 45 years of age are likely candidates for biopsy. Detection of more than 5% fat in cells in the biopsy is sufficient for the diagnosis of NAFLD. When this is accompanied by liver cell destruction and fibrosis, the term NASH is used. If inflammation and fibrosis cannot be prevented, liver cirrhosis will be inevitable.[20-23]

Many agents have been tried in the treatment, but the desired results have not been achieved. The only method that is accepted as effective is to achieve weight loss. Weight loss of 0.5-1 kg per week is ideal.[22] In addition, patients should be vaccinated against viral hepatitis. Patients should avoid excessive alcohol consumption. Patients are mostly lost due to cardiovascular diseases. Therefore, glycemia, blood fat levels, and arterial blood pressure should be kept within the appropriate range. Many different drugs, from anti-diabetic agents to statins, have been tried in these patients, but it has been concluded that only Vitamin E and omega-3 fatty acids may be of limited benefit.[23] Therefore, alternative treatment methods are important.

Chronic Viral Hepatitis

The term hepatitis is used to describe inflammation of the liver. It can develop due to bacterial, viral, and parasitic infections, as well as secondary to alcohol use, drugs, and metabolic diseases. Viral agents such as hepatitis A, B, C, E, D, EBV, and CMV can cause liver damage. However, in this section, when we consider the prevalence, we will mention the factors of chronic viral hepatitis (CVH), namely Hepatitis B, C and D. Chronic hepatitis is a definition used for conditions where inflammation in the liver lasts longer than six months. Effective treatment is very important in CVH because it causes cirrhosis and liver cancer in the long term and creates a reservoir for transmission. It is thought that around half a billion people worldwide suffer from CVH.[24]

All three factors are transmitted by blood, sexual intercourse, and from mother to baby during pregnancy through the placenta. Hepatitis C and D virus is RNA virus, hepatitis B is DNA virus. In those who are infected with hepatitis B, the probability of becoming chronic is one in twenty. It is almost inevitable in newborns who pass through the placenta if they are left untreated. Hepatitis C tends to become chronic at a rate of 65%. After the transmission, there is a period when signs and symptoms are not observed, but the virus is detected by laboratory tests. Then comes the stage characterized by nausea, vomiting, loss of appetite, muscle and joint pain, and weakness. This process is followed by jaundice and a recovery period. On the other hand, very few of the patients diagnosed with CVH show signs and symptoms. They may describe weakness, malaise, and mild pain in the upper abdomen. However, these symptoms are not specific to hepatitis and are often ignored. Tenderness and marginal irregularity may be detected in the liver. Patients are mostly diagnosed because of tests performed for another reason. Rarely, skin rash, arthritis, and renal dysfunction may develop.[24-26]

Approximately one-fifth of patients followed up with chronic hepatitis B develop cirrhosis or liver cancer in the future. The development of liver cancer without cirrhosis distinguishes hepatitis B from other diseases. It is the primary cause of liver cancer. Therefore, annual follow-up with ultrasound and serum α-fetoprotein is recommended for patients older than 45 years of age and with a positive family history of liver cancer.[26-28]

Hepatitis C is almost completely asymptomatic. However, in infected individuals, the disease is most likely to become chronic. One-fifth of these people develop cirrhosis. The incidence of liver cancer after cirrhosis is one in twenty per year.[25,27]

Hepatitis D is a defective virus. It can cause disease only in the presence of a hepatitis B surface antigen. While it can be transmitted simultaneously with hepatitis B, it can affect patients who were previously positive for hepatitis B later. Its course is not different from hepatitis B infection alone. Only, the probability of cirrhosis and liver cancer is six in ten, and the development process is faster. In diagnosis, virus-specific antibodies, liver function tests, viral genome determination, and liver biopsy can be used to determine the differential diagnosis and degree of disease. The main goal of treatment is to reduce the inflammatory process and fibrosis in the liver, thereby preventing cirrhosis and other complications. For this purpose, interferon, immunoglobulin, nucleoside and nucleotide analogs, protease inhibitors, and steroids are used. For protection purposes, it is recommended to vaccinate especially healthcare workers, sex workers, dialysis patients, intravenous drug users, patients with organ transplants, and patients whose immune system is suppressed.[25,28]

Cirrhosis

Cirrhosis is derived from the Latin word cirrus, meaning yellow brown, because of the liver appearance observed at autopsy. It is one of the most feared consequences of long-term liver diseases. Regardless of the underlying cause, progressive fibrosis and regeneration nodules cause the deterioration of the architectural structure of the liver, which is indispensable for its functions. Unfortunately, it is irreversible, except in a few rare cases. It is thought to be responsible for approximately 1% of all deaths.[29] Numerous diseases can cause cirrhosis, either by causing long-term inflammation of the liver or by impairing bile flow. However, chronic viral hepatitis, NAFLD, and alcohol use are the main causes. Genetic disorders of iron and copper metabolism, autoimmune diseases involving the liver parenchyma and biliary tract, infections, and heart diseases can be counted among other causes.[30]

Under normal conditions, the production and destruction activities in the extracellular space continue in a balanced way. However, in inflammatory processes where this balance is removed, molecules that provide communication between the secreted cells stimulate the satellite cells in the perisinusoidal area. These cells also synthesize excessive amounts of collagen and secrete it into the intercellular space. Thus, the sieve-shaped and highly permeable vascular structure of the liver, which provides the relationship between cells and blood, is greatly impaired. In a sense, the communication windows are walled and the blood pressure in the vessel rises. The blood coming from the intestines and to be filtered in the liver accumulates due to congestion and tries to reach the heart by alternative routes. As a result of this, the accumulation of fluid, which we call ascites, occurs between the abdominal membranes, varicose veins occur with the expansion of the vessels between the internal organs, and changes occur in the state of consciousness due to the blood passing to the brain without being cleaned in the liver. This situation, which we are trying to explain simply, is called portal hypertension. The elucidation of these physio pathological mechanisms is very important in terms of providing a horizon for new treatment methods.[30-32]

Patients may present with non-liver-specific complaints such as loss of appetite, weight loss, weakness, and fatigue. Therefore, these patients are often not diagnosed in the early stages of their disease. However, if the disease progresses, more serious conditions such as jaundice, itching, swelling in the abdomen, bleeding from the mouth or anus, and changes in consciousness may occur. Less frequently, women may apply to the hospital with menstrual irregularities and men with impotence.[32] When detailed physical examination is done in patients; jaundice on the skin, reticulated enlargements in the superficial veins,[33] enlargement in the breast can be detected. Abdominal swelling due to fluid accumulation between the abdominal membranes,[34] increase in liver size and irregular margins, enlargement of the spleen, redness of the palm, and changes in nail structure may also be detected.

Laboratory examinations may reveal low hemoglobin, thrombocyte, and albumin levels, prolongation of prothrombin time, and elevated liver enzymes. If ultrasonography, tomography, or magnetic imaging is performed in these patients, nodular structure, marginal irregularity, atrophy, or left-sided hypertrophy can be observed in the liver. In patients with cirrhosis symptoms, if laboratory and imaging findings are supporting this, or if life-threatening complications such as ascites, esophageal variceal bleeding, and liver-related changes in consciousness have developed; biopsy is not necessary for diagnosis. This procedure should be reserved for patients for whom the detailed examination is made for the cause after the diagnosis of cirrhosis, but for which no conclusion can be drawn.[35]

After the diagnosis of the disease is finalized, it is very important to reveal the cause. Because with the interventions to be made, the course of the disease may slow down.[36] However, there are precautions to be taken for all patients. All patients should stay away from alcohol and substances harmful to the liver and be vaccinated against hepatitis B and hepatitis A. Diet and exercises should be regulated and followed, and daily weight measurements should be made. Patients should be advised not to be constipated. They should be followed up with endoscopic imaging for variceal bleeding every two years, and ultrasound and blood tests for liver cancer every six months. Patients should be included in the liver transplant program unless there is a contrary situation in terms of transplantation. However, the high prevalence of the disease, the low number of donors, and the inadequacy of experienced centers prolong the waiting period. In this respect, alternative treatment methods and perspectives are of vital importance for both patients who are not suitable for transplantation and for patients waiting for transplantation.[33-36]

CHRONIC PANCREATITIS

The pancreas is located behind the stomach wall. It acts as the conductor of the metabolism in the digestion and absorbability of food with the enzymes it produces as an exocrine gland and the hormones it secretes as an endocrine gland. Chronic pancreatitis is a process in which the structural integrity of the pancreas is irreversibly impaired due to progressive inflammation and fibrosis and the pancreas cannot perform its functions.[37,38]

Although the incidence varies in societies depending on alcohol consumption habits, it can be said that the annual number of new cases is 3-9/100000. Alcohol, genetic diseases such as cystic fibrosis, conditions that obstruct the pancreatic duct such as stone or tumor, autoimmune diseases, and excessive serum triglyceride levels, may cause chronic pancreatitis. However, although it has been revealed that these diseases are associated with chronic pancreatitis, the development process of the disease is still unknown. One of the hypotheses is that a plug is formed in the pancreatic duct because of a protein-containing secretion that cannot be buffered by secretin and bicarbonate. The presence of patchy inflammatory cells in the autopsy series supports the opinion that immune response occurs in individuals with a genetic predisposition to an unidentified factor or factors, and this process causes long-term damage to the pancreatic tissue. No matter how the process progresses, the unchanging situation is the deterioration and irreversible destruction of the microarchitecture of the organ - as described in cirrhosis - because of increased collagen accumulation in the intercellular space in response to inflammatory cytokines.[38-42]

So, with which complaint do patients apply to the hospital more? -Pain. This pain is localized to the upper midpoint of the abdomen and may radiate to the back. It usually starts 15-30 minutes after meals. It may be accompanied by nausea and vomiting. The patient can relax by sitting and leaning forward. While the pain lasts for a few days in the initial stages of the disease, the pain becomes chronic as the process progresses.[40] Considering that ten percent of healthy tissue is sufficient for the organ to perform its functions, patients may rarely present with pancreatic insufficiency findings. Fatty, foul-smelling stools, weight loss, and deficiency of especially fat-soluble vitamins A, D, E, K, and cobalamin can be observed.[41] Similarly, diabetes may develop because of the destruction of islet cells. Unfortunately, there is no physical examination finding specific to the disease, but tenderness can be detected in the epigastric area.

There is no specific laboratory test that can detect the disease directly. Serum amylase and lipase tests, which are very important in acute pancreatitis, are not very useful in these patients. For this reason, pancreatic ultrasound, tomography, and magnetic imaging can be performed in patients clinically compatible with chronic pancreatitis. Atrophy, duct enlargement, calcification, cyst formation, or anatomical disorder may be observed in the gland. In cases where these examinations are insufficient, endoscopic methods can be tried. In addition, stool fat determination and pancreatic stimulation tests can be performed for malabsorption.[42-44]

Providing pain control is the primary goal in the treatment of the disease. In addition to specific treatment methods for the underlying disease, there are also precautions that all patients must comply with. Smoking and alcohol use should be stopped, and frequent and less nutrition should be provided. A diet rich in medium-chain fatty acids is another method that may be beneficial. In addition, pancreatic enzyme supplements and vitamin preparations can be used. Tricyclic antidepressants, morphine derivatives, and pregabalin can be preferred for pain control. Nerve blockade for the celiac ganglion can be applied in patients who do not benefit. In cases where all these methods are insufficient, surgical methods can be considered.[43,44]

IRRITABLE BOWEL SYNDROME

Irritable bowel syndrome (IBS), which was called spastic colon in the past and is also known as sensitive intestine today, is a common gastrointestinal disorder characterized by chronic abdominal pain and changes in bowel habits. While its incidence is 10-15% (45-50) in North America, its incidence in Europe is around 11.5%, although it varies from country to country. Although IBS can be seen in all ages and genders, it is more common in young women.[45-52]

The pathophysiology of the disease has not been fully elucidated. Traditionally, gastrointestinal motility and intestinal hypersensitivity have been emphasized. Recent studies focus on the role of inflammation, microbiota, food sensitivity, and genetic predisposition.[45,47,51,52]

IBS patients may present to the hospital with a wide variety of symptoms, both gastrointestinal and non-gastrointestinal. Symptoms such as abdominal bloating, flatulence, belching, gastroesophageal reflux, nausea, dysphagia, and early satiety are common in patients with IBS. Although symptoms related to chronic abdominal pain and changes in bowel habits are not only seen in IBS, but they are also necessary for the diagnosis of IBS.[51-53]

CHRONIC ABDOMINAL PAIN

Abdominal pain is described as a cramping sensation of varying severity that flares up from time to time. The location and character of the pain are very variable. Various factors such as emotional stress, and food consumption can increase the pain, defecation usually provides some relief. This pain does not prevent falling asleep and does not wake up from sleep. Abdominal pain should not be associated with weight loss, rectal bleeding, or anemia and should not be progressive.[47,53,54]

Changes in Bowel Habits:

Patients with IBS present with diarrhea, constipation, both diarrhea, and constipation, or a bowel habit that can turn into diarrhea or constipation without both. Diarrhea usually occurs in the form of small to medium-volume soft stools, at the time of waking up in the morning or after meals. Approximately half of the patients have mucus discharge with feces. Large-volume diarrhea, bloody stools, oily stools, and nocturnal diarrhea are not related to IBS and an underlying organic cause should be investigated. Constipation can last for days or months. Defecation is difficult and patients may not feel complete relief after defecation, even if the rectum is empty.[52-54]

IBS is divided into 4 subgroups according to changes in bowel habits.[54,55]

i. IBS with diarrhea
ii. IBS with constipation
iii. Mixed type IBS
iv. Unclassifiable IBS

There is no biological marker or imaging method for the diagnosis of IBS. Considering the symptoms, it was tried to establish standard criteria for diagnosis and the Rome IV diagnostic criteria, which were renewed in 2016, came into use. According to this; diagnostic criteria were defined as the presence of abdominal pain that started at least six

months ago and recurring at least once a week in the last three months, along with two or more of the following three criteria.[55]

i. Relief with defecation
ii. Accompanied by a change in stool frequency
iii. With a change in stool shape

Tests to diagnose the disease should be limited. Further investigation is not recommended if there are no alarm symptoms and the diagnostic criteria for IBS are met. Many conditions such as inflammatory bowel disease, celiac disease, lactose intolerance, fructose intolerance, and microscopic colitis can be confused with IBS because they have symptoms like IBS. Further imaging studies and/or endoscopic examination may be required in patients with alarm symptoms.[53-55]

Since the pathophysiology of the disease is not fully understood, there is no effective treatment method. In the treatment of all patients with IBS, it is important to first establish a reliable doctor-patient relationship. Lifestyle changes and dietary adjustments are recommended for mild IBS patients whose quality of life is not impaired. It is recommended not to consume gas-inducing foods (beans, onions, cabbage, raisins, bananas, apricots…), alcohol, and caffeine. A low FODMAP (fermentable oligosaccharides, disaccharides, monosaccharides, and polyols) diet is recommended. Fiber consumption is recommended in IBS patients with constipation. In moderate and severe IBS patients, symptomatic pharmacological agents are used for the most severe complaints of the patients. These agents can be listed as antidiarrheal drugs, bile acid sequestrants, 5-HT3 receptor antagonists, osmotic laxatives, lubiprostone, spasmolytic agents, antidepressants, antibiotics, and probiotics.[51,53-55]

CONCLUSION

In this review we shortly reported the most common causes of GIS diseases in terms of clinical findings and mostly used treatment modalities. Other aspects of treamentent modalities are in development process and we believe that they will provide many opportunities to clinicans and patients.

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